BAILEY (P.) & EWING (J.)

A CONTRIBUTION TO THE STUDY OF ACUTE ASCENDING (LANDRY'S) PARALYSIS

BY

PEARCE BAILEY, A. M., M. D.,

ASSISTANT IN NEUROLOGY, VANDERBILT CLINIC;
ATTENDING PHYSICIAN TO THE
ALMSHOUSE, WORKHOUSE, AND INCURABLE HOSPITALS, NEW YORK,

AND JAMES EWING, A. M., M. D.,

TUTOR IN HISTOLOGY, COLLEGE OF PHYSICIANS AND SURGEONS,
COLUMBIA UNIVERSITY, NEW YORK;
ASSISTANT PATHOLOGIST TO ROOSEVELT HOSPITAL.

REPRINTED FROM
THE NEW YORK MEDICAL JOURNAL
JULY 4 AND 11, 1896

NEW YORK
D. APPLETON AND COMPANY
1896

A CONTRIBUTION TO THE STUDY OF ACUTE ASCENDING (LANDRY'S) PARALYSIS

BY

PEARCE BAILEY, A. M., M. D.,

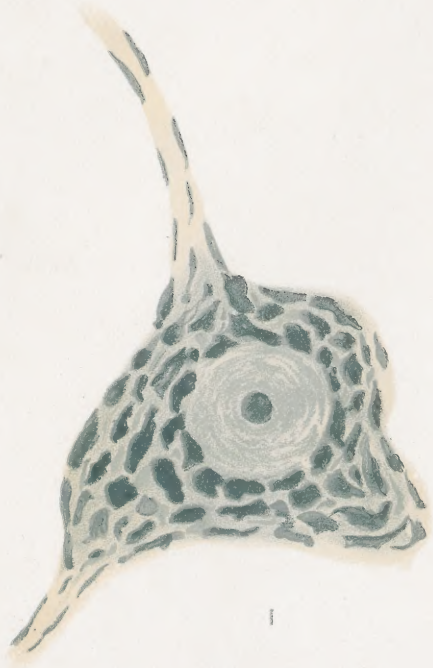
ASSISTANT IN NEUROLOGY, VANDERBILT CLINIC;
ATTENDING PHYSICIAN TO THE
ALMSHOUSE, WORKHOUSE, AND INCURABLE HOSPITALS, NEW YORK,

AND JAMES EWING, A. M., M. D.,

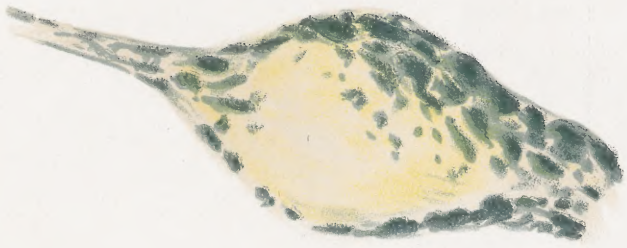
TUTOR IN HISTOLOGY, COLLEGE OF PHYSICIANS AND SURGEONS,
COLUMBIA UNIVERSITY, NEW YORK;
ASSISTANT PATHOLOGIST TO ROOSEVELT HOSPITAL.

REPRINTED FROM
THE NEW YORK MEDICAL JOURNAL
JULY 4 AND 11, 1896

NEW YORK
D APPLETON AND COMPANY
1896

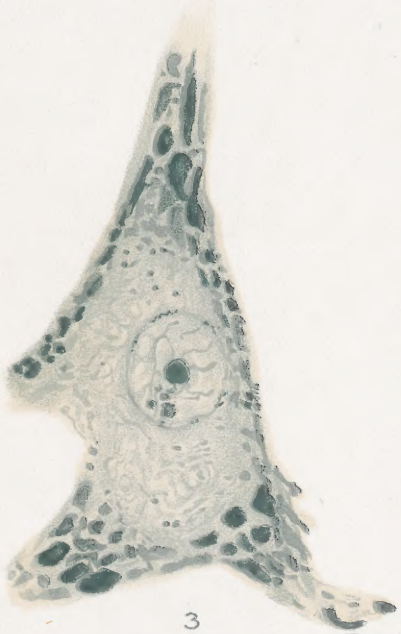


1



2

4



3

5

DR. PEARCE BAILEY'S AND
DR. JAMES EWING'S ARTICLE ON ACUTE ASCENDING (LANDRY'S) PARALYSIS.

Fig. I.—Normal human ganglion cell of the anterior horn of the spinal cord.

Fig. II.—Increase of pigment in the senile ganglion cell.

Figs. III, IV, and V.—Degenerative changes in the ganglion cell.

A CONTRIBUTION TO THE STUDY OF ACUTE ASCENDING (LANDRY'S) PARALYSIS.*

ALTHOUGH it is nearly forty years since the French physician Landry described the disease which bears his name, and although since that time a considerable number of cases of this disease have been reported in different parts of the world, there are to-day many conflicting views of the pathology of Landry's paralysis.

The symptoms of the original case were an acute paralysis, ascending from the legs to the arms, and soon involving the bulbar muscles. The paralysis was unaccompanied by marked loss of sensation, nor did it involve the sphincters. The paralyzed muscles retained their faradaic excitability; the intellectual faculties remained unimpaired and fever was not prominent.

But after death no lesions were found in the nervous system to explain these rapidly fatal symptoms. Although the nerves were not examined, the results of a careful microscopic investigation of the spinal cord by several observers were absolutely negative.

Since that time some cases have occurred which were identical, both clinically and pathologically, with the original, and others have been reported in which more or less serious pathological alterations were found in the nervous system.

A study of the literature of this subject, together

* Read before the New York Neurological Society, March 3, 1896.

with the recent examination of a case of acute ascending paralysis in which the clinical symptoms were in exact agreement with Landry's description, but in which extensive lesions were found in the spinal cord, has led us to certain conclusions which are at variance with some recent conceptions of the pathology of the disease.

The object of the present paper is therefore twofold:

I. The description of a case of acute ascending paralysis, with special reference to its anatomical lesions.

II. A consideration of the cases of Landry's paralysis hitherto recorded, with a view of determining the pathological nature of the disease.

I.—The patient, M. S., a woman, aged thirty-six, housewife, born in Ireland, was admitted to Roosevelt Hospital November 25, 1895, during the service of Dr. Delafield, to whose courtesy we are indebted for the following history:

Family history negative. Personal history: Considerable beer habit; never had articular rheumatism; denies all symptoms of syphilis and shows no evidences of this disease; has always been healthy. Present illness began on November 19th with an attack of vomiting without apparent cause, although she acknowledges a moderate but not unusual indulgence in beer on the previous day. Vomiting continued, till on the third day (November 22d) she went to bed exhausted. At this time she began to have occasional attacks of vertigo and dimness of vision, and on attempting to rise from bed fell to the floor in syncope. On November 23d she rather suddenly lost power in both legs, and on November 24th there was paralysis of the left arm with paresis of the right arm. Sensation was undisturbed.

Condition on Admission.—Color and nutrition good; tongue dry, slightly coated; pulse, 100, regular, tension normal; temperature, 101.4°; respiration, 28, regular; urine, acid, specific gravity, 1.030; no albumin; no sugar; a few hyaline casts. Physical examination of thoracic and abdominal viscera negative.

Nervous System.—Motion: Complete inability to move either legs or left arm; can flex toes of both feet perceptibly; retains slight power to move right arm. There is moderate left ptosis.

Sensation: Tactile sensibility normal; states accurately when and how legs are moved; no pain or tenderness in arms or legs.

Bladder and rectum: Control normal. Reflexes: Lost.

Electricity: Strong faradaic current elicited a diminished response in paralyzed muscles (November 28th).

November 26th.—Vomited repeatedly; sleepless.

27th.—Complained of headache and dyspnœa.

29th.—Urine, fifteen ounces; contains a trace of albumin; no casts. Dyspnœa increased rapidly. Dysphagia noted in the morning, which later became complete. Dysphonia marked and increasing. In the evening the patient became unconscious, and at 8.45 P. M., with severe dyspnœa, cyanosis, and pulmonary œdema, she died.

Autopsy (Dr. Ewing).—November 30th, twenty hours after death. Body fat, pale; no œdema or noteworthy external markings.

Serous cavities: Normal. A few old adhesions over left lung.

Lungs: Moderately congested, decidedly œdematous; bronchi slightly reddened and coated with frothy mucus; bronchial lymph nodes normal.

Heart: Size, surfaces, chambers, and valves normal; muscle rather pale; aorta presents a very few atheromatous patches.

Liver: Moderately enlarged; consistence much reduced; surface and section smooth; centres of lobules deep red, not depressed; peripheries light-colored and fatty, but general outlines of lobules indistinct.

Spleen: Decidedly enlarged, rather firm, section deep red, trabeculæ faintly visible; Malpighian bodies indistinct.

Kidneys: Moderately enlarged, soft; capsule not adherent; surface smooth; stellate veins congested; cortex slightly thickened and congested; markings regular; pyramids much congested.

Gastro-intestinal tract: Adrenals, ureters, and pancreas apparently normal (Peyer's patches not enlarged).

Muscles: Not examined.

Brain: Considerable distention of veins and sinuses of brain and dura. No thrombi. Pia and cortex rather succulent. Sections through cortex, capsules, basal ganglia, medulla, and pons disclosed a large but not distinctly abnormal blood content; no gross lesions.

Spinal cord: There was the usual filling of the dural

veins. Pia normal. Consistence of cord normal. On section, the white matter throughout seemed entirely normal. The gray matter appeared plainly outlined and the vessels slightly congested, but these features were not more prominent than is frequently found in normal cords. At one point in the gray matter of the cervical cord a minute red spot was found which could not be displaced, indicating the presence of extravascular blood.

Anatomical Diagnosis.—Congestion and œdema of the lungs; acute degeneration of liver and kidneys; acute hyperplastic splenitis.

Diagnosis of the condition of the nervous system reserved for microscopical examination.

The brain and cord were placed in Lang's fluid * for forty-eight hours, the basal ganglia having been separated by Meynert's method, and the cord having been divided by transverse incisions at short intervals. The specimens were then washed in water and treated with successive alcohols of increasing strengths, tinged with iodine to remove all trace of sublimate, and finally mounted in celloidin and sectioned.

The stains employed were the eosin hæmotoxylin, Van Gieson's picro-acid fuchsine, Loeffler's alkaline methylene blue, Ehrlich's tri-acid mixture, Gram's and Nissl's.

The Nissl stain furnished the most satisfactory results and was the one chiefly employed. This stain is to be most highly recommended for the study of changes in the ganglion cells. Not only is its application simple, but it demonstrates better than any other method the minute structure of the cell.

After Nissl's method, the sections were gently heated in a one-per-cent. aqueous solution of methylene blue over a small Bunsen burner. They were then washed in water, decolorized, and at the same time dehydrated in strong alcohol (absolute is preferable), cleared in oil of origanum, and mounted in balsam.

Microscopical Examination.—Cauda equina: The vessels of the cauda equina were filled with blood, but there was no thickening of their walls or infiltration of the circumvascular sheaths. The axis cylinders of the nerve fibres appeared normal, and there was no increase of the connective-tissue nuclei.

Nerve roots: The nerve fibres, so far as could be de-

* Distilled water, 2,000; chloride of sodium, 120; acetic acid, 120; bichloride of mercury, 60.

termined by the methods used, were normal. There was no increase of the nuclei of the neuroglia cells. In some of the nerve roots there was a slight circumvascular infiltration of small round cells.

Pia mater: There was some round-cell infiltration in the meshes of the spinal pia, especially about the vessels.

Peripheral nerve trunks were not examined.

Spinal cord—gray matter: The gray matter was extensively affected throughout the entire length of the cord, being much more involved in some areas than in others. The lesion consisted in intense congestion of all the blood-vessels, especially of the ramifications of the central branch of the anterior spinal artery. In the cervical region there were several capillary hæmorrhages, and some of the arterial twigs contained thrombi composed of multinuclear leucocytes. Nearly all the vessels showed a pronounced circumvascular infiltration with small round cells (Fig. 1). In some places this infiltra-

Fig. 1.—Circumvascular infiltration of small round cells. Vein of anterior horn.

tion had extended beyond the circumvascular sheath, and the small cells were gathered in such numbers as to form what may be properly regarded as miliary abscesses. Furthermore, there was a diffuse cellular infiltration in the gray matter, which sometimes extended slightly

into the white matter. At some levels congestion and exudation were so intense that nothing could be distinguished but the round-cell infiltration and the detritus of neuroglia and ganglion cells. Where the process had extended into the white matter, as was observed in the cervical region only, the fibres were absent and their places occupied in part by large mononuclear cells, with homogeneous or granular protoplasm.

The central canal appeared not specially affected.

Before detailing the degenerative processes in the ganglion cells, it may be well to briefly describe a normal human ganglion cell of the anterior horn as it appears when stained by Nissl's method (Fig. 1, Plate). The cell body under these conditions contains a number of chromophilic masses, larger and grouped concentrically about the nucleus, arranged in rows along the cell borders, and prolonged as slender rods into the dendrites. These bodies are not found in the axis-cylinder process.

The significance of these chromophilic bodies is not known, but they are believed to be a characteristic and normal element of the ganglion cell. Besides these bodies smaller granules are scattered through the cytoplasm, giving the appearance of a fine network. Some areas of diffuse, structureless, greenish pigment are often encountered in normal ganglion cells of the adult, a feature which is much more conspicuous in old age (Fig. 2, Plate). By this method of staining the nuclear membrane is distinct, and is separated from the deeply staining nucleolus by a clear area in which may occasionally be made out a fine granular network.

The abnormities in the ganglion cells observed in the present case consisted principally in the partial or complete absence of the chromophilic masses (Figs. 3 and 4, Plate). In cells in which the lesion was least marked the absence of chromophilic bodies was noted only in the central portions of the cells. Other cells were seen in which the cytoplasm was entirely bereft of chromophilic bodies and their places were occupied by a multitude of fine, rather deeply staining granules, which gave to the cell body a very peculiar "dusty" appearance. Further, in many cells even the finer bluish particles

were absent, and in these cells especially, less often in the others, the nuclear membrane was indistinct, irregular, or granular, and the nucleolus fragmented or absent (Fig. 5, Plate).

Finally, in the areas where evidences of exudative inflammation were most pronounced, many shrunken and irregular cell bodies and collections of deformed chromophilic bodies without visible cell nuclei were with difficulty to be recognized.

The fragmentation of cell processes, as observed by Oettinger and Marinesco, was occasionally encountered, but in such situations as to render its pathological significance uncertain.

While not insisting that our interpretation is entirely correct, we believe that the real chronological order of these degenerative changes in the ganglion cells has been indicated by the sequence of the above description.

White matter: Besides the few areas where the inflammatory process in the gray matter had extended into it, the only lesion of the white matter was a moderate circumvascular infiltration of the blood-vessels.

The Topographical Distribution of the Lesion in the Cord.—In the cervical region the lesion was most pronounced, and in many sections not a single ganglion cell had escaped extreme alteration or complete destruction.

In the lumbar and dorsal regions (Fig. 2) the changes were extensive, but the cells of Clarke's column were only slightly affected, and offered a sharp contrast to the intensely inflamed anterior horns.

In the sacral region and conus medullaris the lesions were least pronounced, and many ganglion cells in the anterior horns remained in a fair condition of preservation.

The changes in the gray matter throughout the entire cord were but slightly marked in the posterior horns.

In the cervical region only, as before mentioned, some fibres of the white columns had suffered destruction by an extension of the inflammatory process from the gray matter.

The nerve roots in the upper dorsal and cervical segments showed considerable circumvascular infiltration, but their fibres were well preserved. Although the peripheral nerves were not examined, it is reasonable to suppose that any lesion which may have existed in them was secondary to the intense inflammation of the spinal cord.

Medulla and brain: The lesions in the medulla and pons were chiefly marked in the gray matter along the

floor of the fourth ventricle and in that of the pons, although the white matter and periphery were not entirely uninvolved. The changes were similar to those described in the cord. The nuclei of all the cranial nerves were more or less damaged, those least affected being the sixth and seventh. The changes in the nuclei of the third nerves were chiefly in the ganglion cells which lie

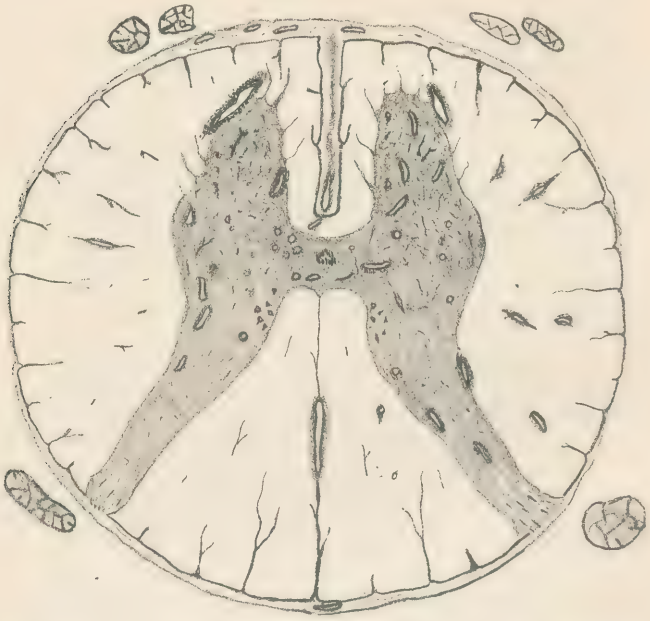


FIG. 2.—Section of spinal cord—dorsal region. Ganglion cells of Clarke's column only preserved.

internally. The nucleus funiculi teres was but little affected. The basal ganglia showed the circumvascular lesions and numerous small foci of cellular infiltration. The internal capsule was intact, but the motor cortex presented the same small collections of round cells, and ganglion cells in the milder stages of degeneration. The temporo-sphenoidal lobes were normal. The marked lesions of the medulla extended slightly into the middle peduncles and the lateral lobes of the cerebellum.

Throughout the cerebellar cortex (Fig. 3) were found lesions similar to those seen in the spinal cord. The layer of Purkinje's cells was in many of the convolutions dis-

placed outward from the nuclear layer, and in adjoining inflammatory foci some of these cells were absent.

No bacteriological examination was made either by culture or by animal inoculation.

FIG. 3.—Lesion in cerebellum.

Sections from various levels were stained by Loeffler's alkaline methylene blue and by Gram's method, but no bacteria were found.

Summary.—The foregoing case furnishes an exact clinical counterpart of the one originally reported by Landry, and presents symptoms regarded by subsequent authorities as essential in this disease—viz., an acute ascending paralysis, causing death by invasion of the bulbar nuclei, without marked sensory impairment, with-

out involvement of the bladder or rectum, and with retained faradaic excitability of the muscles.

But, in contrast to his case and to a few subsequent well-authenticated examples of this disease, in the present instance the clinical symptoms are attributable to most extensive lesions, involving the whole cerebrospinal axis, affecting chiefly the gray matter of the brain and medulla and the anterior horns of the spinal cord.

The character of the lesion was that of an acute exudative inflammation, with marked cellular infiltration of the circumvascular sheaths, degeneration of the ganglion cells, and obliteration of other structural elements.

That these lesions are but slightly marked in the sacral region will explain the integrity of the sphincters, and the fairly normal condition of the posterior horns, the sensory tracts, and the nerve roots accords with the absence of sensory symptoms.

II. Consideration of the Pathology of Acute Ascending Paralysis.

The literature which has grouped itself around the association of symptoms known as Landry's, or acute ascending paralysis, is considerable. Many cases have been assigned to this category which manifestly belong to simple polyneuritis; still others have been described which were nothing more than the ordinary disseminated or transverse myelitis. Some cases of general myelitis of rapidly ascending type seem partially to resemble the disease under discussion, and it is at present impossible to state whether or not they should properly be classed as Landry's paralysis. Such a case of ascending myelitis, of seven weeks' duration, which proved fatal, with evidences of involvement of the bulbar nuclei, has been reported by Schultz and Schultze (*Arch. f. Psych.*, 1881, xii, p. 457).

Two papers appeared in 1889 (Nauwerck and Barth, Ziegler's *Beiträge*, 1889, v, p. 1; Ross, *A Treatise on Peripheral Neuritis*, London, 1893, p. 16) which essayed to prove that the disease was essentially a neuritis. But,

in support of this view, cases were included whether accompanied or not with satisfactory pathological reports. Such a view of the pathology of Landry's paralysis was not satisfactory to Hun, who, in a paper two years later, concluded that "acute ascending paralysis, defined so as to exclude all cases in which the sensory symptoms are prominent, or in which marked bulbar symptoms are not present, must be regarded as a clinical entity for which no corresponding lesion has as yet been discovered." This view of Hun's is indorsed by Osler (*Practice of Medicine*, New York, 1895).

After a careful review of all the literature relative to this disease, we have been forced to the conclusion that this opinion will now admit of some modification, as we have found many well-recorded instances of the disease in which the ascending paralysis, unaccompanied by marked sensory symptoms, soon involved the bulbar nuclei, and in which adequate pathological changes were found in the nervous system. For the purpose of classification, we have arranged a table of cases of acute ascending paralysis which have run a fatal course.

Believing that satisfactory conclusions can be drawn only from those cases in which the report was clear on the essential clinical symptoms, and in which a microscopic examination of the nervous system had been made, only a small proportion of the cases reported as Landry's disease have been included in our tables. A few cases have been disregarded on account of clinical deficiencies, many because of the lack of proper pathological examination. The futility of considering cases in which the nervous system was not carefully examined microscopically will be evident to all who are familiar with the technical problems of nervous pathology. We may cite as an example our own case, in which with extensive lesions the spinal cord presented no changes which the naked eye could detect.

If, however, we are to enlarge the conception of Landry's paralysis, as suggested by Nauwerck and Barth, so as to include cases in which sensation was decidedly affected or completely lost, and in which the disease

was distinctly subacute or chronic, with intervals of improvement, the doors are thrown open to a multitude of cases of neuritis, and Landry's paralysis becomes a disease of frequent occurrence and heterogeneous character.

An ascending paralysis which pursues a rapidly fatal course without prominent sensory symptoms is a clinical entity too distinct to warrant, without more definite reasons, such a widening of the conception of this disease. And while the strict adherence to a distinct clinical type may exclude cases in which, although the symptoms are not identical, the morbid process is essentially the same, the present lack of knowledge as to what Landry's paralysis really is renders such conservatism imperative.

The accompanying tables contain, therefore, all the reported cases of Landry's paralysis in which, in our judgment, the results of satisfactory clinical and pathological examination have been given. The references to the whole literature, from which the cases have been selected, may be found in the papers of Nauwerck and Barth and of Ross, and in reports which have appeared subsequently and up to the present time.

We have been unable to study cases published only in the Russian or Scandinavian languages or to obtain certain old monographs. With these exceptions, we believe that our review of the literature has been complete.

A detail of all the cases reviewed would render this paper unnecessarily cumbersome; we have appended, however, a list of some of the more important cases which do not appear in the tables, although a microscopic examination was made.*

* The following cases do not appear in the tables because the clinical history was deficient, or the symptoms and course of the disease were not characteristic, or the pathological report was not complete:

Bernhardt. *Zeit. f. klin. Med.*, 1886, p. 363. Course of disease irregular. Sensation much affected.

Brochain. *Gaz. des. hôp.*, 1854, p. 93. Anæsthesia.

Bourdillat. *Gaz. des hôp.*, 1868, p. 5. Clinical history incomplete.

Buck. *Lancet*, 1895, ii, p. 12. Pathological report incomplete.

In addition, we have included several cases which have hitherto been spoken of, not as Landry's, or acute ascending paralysis, but as acute anterior poliomyelitis. The close clinical adherence of these cases to Landry's

Cornil and Lepine. *Soc. de biol.*, 1873, p. 206. Anæsthesia of feet and legs. General myelitis.

Dejerine. *Compt. rend.*, 1878, v, p. 87, No. 3. No clinical histories.

Diller and Meyer. *Am. Jour. of the Med. Sci.*, 1896, p. 404. Prolonged atypical course.

Eichhorst. Virchow's *Arch.*, 1876, p. 69. Chronic course.

Fox. *Brain*, 1880, ii, p. 418. Unsatisfactory pathological examination.

Gombault. *Arch. de phys.*, 1873, v, p. 80. Duration, five months and a half.

Harley and Clarke. *Lancet*, 1868, ii, p. 451. Specimens examined in fresh condition only.

Klebs. *Deut. med. Woch.*, 1891, xvii, No. 3. No clinical history.

Kussmaul. Reference in Ross. History incomplete. Anæsthesia.

Leudet. *Arch. gén.*, 1865, vi, p. 525. Unsatisfactory pathological report.

Leyden. *Allg. Zeit. f. Psych.*, 1875, p. 537. Two cases. Not typical clinically; lesions in medulla.

Leyden. *Zeit. f. klin. Med.*, 1880, p. 413. Three years' duration.

Lunz and Manurowski. *Abst. Neur. Ctrlbl.*, 1890, ix, p. 696. Clinical history of ordinary alcoholic neuritis.

Nauwerck and Barth. *Loc. cit.* Duration, three months.

Oulment and Hayem. *Gaz. des hôp.*, 1867, p. 405. Subacute course. Sensory involvement from the first.

Pitres and Vaillard. *Arch. de phys.*, 1867, ii, p. 150. Anæsthesia.

Pribytkow. *Revue neurol.*, 1893, p. 672. Total loss of sensibility.

Ross. *Dis. of the Nerv. Sys.*, second edition, vol. i, p. 905. History and pathological report unsatisfactory.

Ross. *Loc. cit.* History incomplete

Roth. *Correspbl. f. s. Aert.*, 1883, No. 13. Paralysis not ascending.

Schultz and Schultze. *Arch. f. Psych.*, 1882, xii, p. 458. Irregular course. Myelitis.

Strumpell. *Arch. f. Psych.*, 1883, xiv, p. 339. Chronic course.

Suydekine. *Ctrlbl. f. klin. Med.*, 1887. Not typical clinically.

Van den Velden. *D. Arch. f. klin. Med.*, 1887, xiv, p. 333. Spastic paralysis.

Vierordt. *Arch. f. Psych.*, 1883, xiv, p. 678. Chronic course.

Westphal. *Arch. f. Psych.*, 1875, vi, p. 765. Case III: Symptoms of neuritis. Nerves not examined. Case IV: Chronic course.

description will readily be seen, and their right to be classed as examples of Landry's paralysis must be admitted. It may be stated also that with few exceptions all the cases given in the tables have been studied from the original papers.

For purposes of classification and comparison, the cases quoted have been divided into three groups:

Group I contains cases in which the result of microscopical examination was negative.

Group II contains cases in which lesions were found —(a) of cord alone, (b) of nerves alone, (c) of cord and nerves.

Group III contains cases which correspond clinically to the type of Landry's paralysis, but which have hitherto been classed as anterior poliomyelitis.

Williamson. *Med. Chron.*, 1890, xii, p. 454. Clinical history indefinite.

Wood and Dercum. *Ther. Gaz.* (Detroit), 1885, p. 157. Case not typical clinically.

Tables compiled from All the Cases found in the Literature of which the Symptoms were those of Landry's Paralysis, and in which Microscopical Examination of the Nervous System was Made.

GROUP I.—*Cases in which no Lesions were found on Microscopical Examination.*

References.	Sex, age, duration.	Ætiology.	Paralysis.	Sensation.	Sphincters.	Reflexes.	Electricity.	Pulse and temperature.	Autopsy.
Landry, *Gaz. héb.*, 1859, t. vi, p. 472.	Male; 43 years; 8 days.	Imperfect recovery from several febrile attacks, with pain in side.	Increasing paralysis successively of legs, arm, trunk, tongue. Death from asphyxia.	Numbness, tingling in fingers and toes for 6 wks. previous; pain not mentioned; anæsthesia of soles of feet and fingers; diminish'd sensation elsewhere in extremities; muscul'r sense intact; special senses normal.	Bladder normal.	Lost.	Normal reaction to faradaism.	P., 90. Slight fever.	No lesion in brain or cord; nerves not examined.

References.	Sex, age, duration.	Ætiology.	Paralysis.	Sensation.	Sphincters.	Reflexes.	Electricity.	Pulse and temperature.	Autopsy.
Pellegrino Levy, *Archiv. général.*, 1865, vi, 1, p. 132.	Male; 22 years; 12 days.	None.	Paresis, followed in 5 days by complete paralysis of legs; later, paresis in arms, dysphagia, asphyxia.	Numbness and tingling in fingers and toes; sensibility to pain and touch preserved; movements caused great pain.	Not mentioned.	Lost.	Not mentioned.	P., 90 to 94. T. not mentioned.	Nerve roots, spinal ganglia, vagi, and spinal cord pronounced normal by Cornil.
Bernhardt, *Berl. klin. Woch.*, 1871, p. 561.	Male; 30 years; 9 days.	Small-pox.	Paresis of legs, later of arms; later paralysis of legs; dysphonia, dysphagia, asphyxia.	Numbness and loss of muscular sense; sensibility to cold and heat retained; pains in joints.	Normal.	Normal.	Normal.	P., 132, regular. T., 99°.	Basal ganglia, medulla, cord, vagi, sciatic, sympathetic, normal; acute hyperplastic splenitis.
Westphal, *Arch. für Psych.*, 1875, v, p. 765.	Male; adult; 28 days.	Phthisis.	Gradual paresis and almost complete paralysis of legs and arms; dysphagia, dysphonia, asphyxia; right pupil paretic.	Moderately diminish'd; muscul'r sense and sensibility to heat and cold intact.	Not mentioned.	Slightly diminished.	Normal.	P., 94, regular. T., 103°.	Cord, medulla, and nerve roots normal; acute hyperplastic splenitis.

Westphal, *loc. cit.*	Male; 32 years; 28 days.	Five weeks after diphtheria.	Paresis, later paralysis of legs and arms; dysphonia, dysphagia, asphyxia.	Much diminished; muscular sense diminished; no pain.	Not mentioned.	Knee-jerks lost.	Normal.	P., 108. T., 100·5°.	Brain, cord, cranial nuclei, nerve roots, ganglia, and muscles normal; acute hyperplastic spelnitis.
Kahler and Pick, *Arch. für Psych.*, 1880, x, p. 313.	Female; 12 years; 11 days.	None.	Paresis, later paralysis of legs and arms; dyspnœa, dysphagia, asphyxia.	Feet anæsthetic; temperature sense delayed.	Not mentioned.	Lost.	Diminished reaction to galvanism.	P., 120. T., 101°.	Acute hyperplastic splenitis; cord normal; adhesions between spinal dura and pia; nerves not mentioned.
Kuemmell, *Zeit. f. klin. Med.*, 1881, ii, p. 273.	Male; 25 years; 6 days.	Fourth week of typhoid fever.	Paresis of arms and legs; paralysis of legs; total paralysis of all extremities; speech thick; double facial paralysis; dysphagia, dyspnœa, asphyxia.	Normal.	Not mentioned.	Normal.	Normal.	P., 80 to 150. T., normal	Bilateral hæmorrhage of medulla (immediately ante mortem?); cord normal; nerves not mentioned.
Strumpell, *Arch. für Psych.*, 1883, xiv, p. 353.	Male; 22 years; 15 days.	None.	Legs, arms; cyanosis, asphyxia.	Pain and tenderness; sensibility normal.	Normal.	Lost.	Not given.	P., 140 at end. T., 103°.	Cord normal; nerves not examined.
Mann, *Med. Chron.*, 1887, vi, p. 99.	Male; 49 years; 9 days.	None.	Legs, arms; dysphagia.	Numbness.	Normal.	Lost.	Retained.	Normal.	Spleen normal; cord, medulla, and brain normal; nerves not examined.
Greppin, *Correspblatt. f. schweit. Aerzte*, 1892, p. 517.	Female; 29 years; 19 days.	Patient insane.	Paralysis of one leg, then of the other; then of one arm, then of the other arm; asphyxia.	Not given.	Retention of urine.	Not tested.	Not given.	No fever.	Brain, cord, peripheral nerves, spinal ganglia, and nerve roots found healthy.

References.	Sex, age, duration.	Ætiology.	Paralysis.	Sensation.	Sphincters.	Reflexes.	Electricity.	Pulse and temperature.	Autopsy.
Ormerod, *St. Bartholomew's Hosp. Rep.*, 1892, xxviii, p. 137.	Male; 20 years; 8 days.	Onset after prolonged swimming.	Paralysis first of legs, then of arms, then of respiration; paralysis of left facial, orbicularis palpebrarum of both sides, and of orbicularis oris; uvula deflected; asphyxia.	Normal.	Normal.	Absent.	Normal.	Normal; pulse not given.	Spleen large, soft; sciatic and vagus normal, stained by Weigert, picrocarmin, aniline blue black; spinal cord, medulla, and cortex normal.
Ormerod, *loc. cit.*	Male; 40 years; 17 days.	Alcoholic. Influenza?	Paraplegia and atrophy; paralysis of left arm, less in right; asphyxia.	No pain, but muscles tender on pressure; formication; sensation impaired in both legs, less affected in arms.	Constipation; difficult micturition.	Reflexes absent in arms; knee-jerk not tested	Normal.	T, 98° to 101·8°.	Nerves and cord stained with osmic acid, Pal, picrocarmin; all normal except in one vagus (Müller's fluid), in which "fibres did not take "Weigert" well, and stained too deeply in carmin."
Ormerod, *loc. cit.*	Male; 33 years; 16 days.	Alcoholic?	Paraplegia, dysphagia.	Impaired in legs and arms; pain in back; girdle sensation.	Incontinence of urine; constipation	Absent.	Not used.	T., 99° to 102·2°.	Spleen large, soft; cord (Müller's fluid) aniline blue black, normal; anterior tibial nerve (Müller's fluid) and cauda equina (osmic acid) both normal.

Watson, *Brit. Med. Jour.*, 1892, ii, p. 1286.	Male; 52 years; 8 days.	After lying on damp ground.	Paraplegia ascending and ending in respiratory paralysis; dysphonia, dysphagia, asphyxia.	No pain or anæsthesia.	No incontinence.	Absent.	Not mentioned.	Not given.	Histology (no details) of cord negative; nerves not mentioned.
Albu, *Zeit. f klin. Med.*, 1893, xxiii, p. 387.	Male; 46 years; about 2 weeks.	None.	First paraplegia, then paralysis of arms; dysphonia, dysphagia, asphyxia.	Normal. "Intense tenderness of whole body," most marked in neck.	Normal.	Absent.	Not tried.	Normal; excessive sweating.	Nerves, cord, and spleen normal; bacteriological examination of cord, nerves, and blood (sowed on agar) negative.
Leube, *Special Diagnose der in. Krank.*, Leipzig, 1893, p. 142.	Female; 26 years; 8 days.	None.	Paralysis of legs and arms; dyspnœa, tachycardia, dysphagia, double facial paralysis; lagophthalmos.	Absolutely intact; pain in back.	Paralyzed at close of illness.	Lost.	Normal.	No fever. P., 150.	Absolutely negative (all specimens in Müller's fluid).

GROUP II (a).—*Cases in which Lesions were found in the Spinal Cord.*

Eisenlohr, *Virchow's Arch.*, 1878, lxxiii, p. 73.	Male; 42 years; 9 days.	Not stated.	Paresis simultaneously of arms and legs; dysphagia, dysphonia, asphyxia.	Nearly normal.	Normal.	Lost.	Normal.	P., 136. T., 98°.	In pons and medulla small foci of round cells about vessels and in tissues; ganglion cells "shining" and "swollen"; capillary hæmorrhages; central canal and vicinity infiltrated with round cells; peripheral nerves normal; spleen, acute hyperplastic splenitis.

References.	Sex, age, duration.	Ætiology.	Paralysis.	Sensation.	Sphincters.	Reflexes.	Electricity.	Pulse and temperature.	Autopsy.
Ross, *Dis. of Nerv. Sys.*, 1883, i, p. 905.	Female; 21 years; 5 days.	Syphilitic.	Paralysis of legs, paresis of arms; later paralysis of all extremities; dyspnœa, asphyxia.	Not materially affected.	Not mentioned.	Not mentioned.	Not mentioned.	Not mentioned.	Disappearance of some ganglion cells; increase of nuclei; dilatation and congestion of blood-vessels.
Roussel, *Arch. de med. nav.*, 1883, xxxix, p. 370.	Male; 39 years; 6 days.	None.	Arms, legs; dysphagia, dysphonia, asphyxia.	Diminished; numbness and tingling.	Normal.	Not given.	Not given.	Normal.	Vascular lesions in cord; degeneration of ganglion cells; nerves not examined.
Hoffmann, *Arch. f. Psych.*, 1884, xv, p. 140.	Female; 36 years; 14 days.	Not mentioned.	Paresis of legs, then of arms; dysphonia, dysphagia, asphyxia; right ptosis; no atrophy.	Formication, but no pain or anæsthesia; tinnitus aurium.	Retention urine at the end.	Plantar reflex normal; knee-jerk lost.	Faradaic reaction diminished in right facial only.	Not mentioned.	Round cells along vessels of meninges and some vessels in "nerve substance," from dorsal cord to upper medulla; more marked in cord; ganglion cells "shining" and "swollen" and nuclei often obscure; small capillary hæmorrhages in cervical and dorsal cord; nerve roots negative.
Immerman, *Neur. Centralb.*, 1885, iv, p. 304.	Male; 22 years; 4 weeks.	None.	Ascending paralysis of lower and upper extremities; bulbar symptoms later; asphyxia.	Normal.	Paretic.	Lost.	Normal.	Moderate fever.	Cellular infiltration of walls of vessels; disappearance of ganglion cells; lesion limited to anterior horns of gray matter of entire cord; peripheral nerves and muscles normal.

Curschmann, 1886, *Verh. der V. Cong. in. Med.*, p. 473.	Male; 31 years; 9 days.	Typhoid fever.	Paresis, later paralysis of legs; paresis of arms, later bulbar signs; slight return of motion in legs day before death.	Tenderness along spine; no other points mentioned.	Normal.	Lost.	"Unsuccessful."	P., 140. T., 104°.	No report on changes in cord except that in white substance were round masses of bacilli which, cultivated and inoculated in rabbits, "proved positively their typhoid nature."
Ketli, *Wien. med. Blatt.*, 1887, x, p. 250.	Male; 30 years; 4 days.	None.	Legs, arms, respiratory muscles.	Unaffected.	Normal.	Diminished.	Normal.	Presumably normal.	The changes in the spinal cord were those of acute anterior poliomyelitis; no other details.
Iwanow, *St. Petersb. med. Woch.*, 1888, v, p. 393 (2 cases).	Age and sex not stated; 8 and 11 days.	None.	Ascending.	Slightly affected in one case only; no pain.	Normal.	One case, not stated; one case, right knee-jerk lost.	Not used.	Pulse not stated; no fever.	Similar changes in both cases; many foci of small round cells regularly arranged along vessels of gray matter or grouped between ganglion cells; destruction of ganglion cells; fibrinlike exudate around vessels; nerves not examined; no bacteria seen by Gram's stain.
Hlava, *Ref. in Schmidt's Jahrbuch*, 1891, 232, p. 244.	Female; 36 years; 4 days.	None.	Sudden loss of power in arms and legs.	Not stated.	Not stated.	Not stated.	Not stated.	Fever.	No gross changes of lumbar cord and corpora quadrigemina; small-celled infiltration, especially in anterior and posterior horns and Clarke's columns; white matter normal; vessels everywhere dilated; cranial nuclei (xii, xi, x) involved; in ulnar and sciatic nerves were found mast cells in the nerve sheaths, but no degeneration.

References.	Sex, age, duration.	Ætiology.	Paralysis.	Sensation.	Sphincters.	Reflexes.	Electricity.	Pulse and temperature.	Autopsy.
Oettinger and Marinesco, *Sem. méd.*, 1895, No. 6.	Male; 20 years; 3 days.	During variola.	Paralysis ascending to arms; asphyxia.	Diminution of all forms of sensation.	Retention of urine.	Lost.	Not stated.	T. that of variola.	Dorso-lumbar cord soft; capillary hæmorrhages; nerves of lower limbs normal; changes in cord follow vascular distribution; vessel walls thick and filled with leucocytes containing basophilic granules; thrombi in some vessels; cocci found in ganglion cells rarely, and in circumvascular spaces and leucocytes frequently; cocci also in central canal; no cultures; nerve cells show degenerative changes; lesion most marked in gray matter; most severe in dorso-lumbar cord, though present in cervical region, medulla, and pons.
Remlinger, *Méd. mod.*, 1896, No. 27, p. 213.	Male; 23 years; 11 days.	Malaria?	Paraplegia; later paralysis of all four extremities; dysphagia, dysphonia, asphyxia.	Pains in leg, worse on movement; no loss of sensibility.	Normal.	Abolished.	Not given.	P., 96. Normal.	Acute hyperplastic splenitis. Spinal cord (Nissl): inflammation in central branch of anterior spinal artery; no thrombi; few streptococci seen in section; spinal cord inoculated in bouillon gave pure culture of *Streptococcus longus*. Injected in rabbits—no effect.

Marie and Marinesco, *Rev. d. sc. méd.*, 1896, 93, p. 134.	Male; 19 years; 7 days.	None.	Ascending to bulbar muscles.	Lost in legs.	Retention of urine.	Not given.	Not given.	Not given.	Softening in anterior horns of cord; hæmorrhages in fourth ventricle; disappearance of nervous elements in anterior horns and infiltration of leucocytes; bacteria in sections resembling anthrax.
Bailey and Ewing (present article).	Female; 36 years; 10 days.	None.	Paralysis of legs, then of right arm, with paresis of left arm; left ptosis; dyspnœa, dysphagia, dysphonia, asphyxia.	Normal; no pain.	Normal.	Lost.	Retained.	P., 100. T., 101·5°.	Acute poliomyelitis of spinal cord and medulla; vascular and exudative changes in motor cortex, basal ganglia, and cerebellum; degeneration of ganglion cells; slight vascular changes in nerve roots; peripheral nerves not examined; acute degeneration of viscera.

GROUP II (b).—*Cases in which Lesions were found in the Peripheral Nerves.*

Dejerine et Goetz, *Arch. de physiol.*, 2d series, t. iii, 876, p. 312.	Male; 45 years; 5 days.	None	Complete paralysis of legs, later of arms; dyspnœa, asphyxia.	Pains in legs and arms; sensation to touch normal.	Retention of urine.	Not stated.	Not stated.	P, 100. T., 102°.	Spinal cord (chromic acid, carmin) found normal; degeneration of fibres, with increase of nuclei in neuroglia, in some of the anterior nerve roots.
Putnam, *Boston Med. and Surg. Jour.*, 1889, p. 159.	Male; 28 years; 7 days.	None.	All four extremities; no bulbar signs until the end, then dysphagia and dyspnœa.	Pains over whole body; numbness of feet.	Not stated.	Not stated.	Not stated	Normal.	Acute hyperplastic splenitis; peripheral neuritis; cord not examined.

References.	Sex, age, duration.	Ætiology.	Paralysis.	Sensation.	Sphincters.	Reflexes.	Electricity.	Pulse and temperature.	Autopsy.
Eisenlohr, *Deut. med. Woch.*, 1890, No. 38, p. 841.	Male; 18 years; 8 days.	Three weeks previously fever and headache.	Paralysis ascending, involving medulla.	Paræsthesia; sensibility and muscul'r sense retained; nerve trunks sensitive to touch; no pain.	Normal.	Lost.	Normal.	Normal.	Blood sterile on gelatin, and microscopically contained no organisms; spleen very large; spinal cord normal; degenerated fibres in right hypoglossal and right phrenic, in anterior roots of cervical nerves, and in other peripheral nerves; no changes in posterior roots; bacterial stains negative in nerves; thorough bacteriological examination of spleen, cord, and nerves negative.
Hun, *N. Y. Med. Jour.*, 1891, liii, p. 609.	Male; 45 years; 12 days.	None.	Paralysis of all extremities and left facial nerve; dysphonia, dysphagia, asphyxia.	Normal; no pain.	Retention of urine.	Absent.	Normal.	Normal. Pulse regular, rapid.	Slight cerebral and spinal meningitis; infiltration of walls of some veins of spinal pia mater, and degeneration of some fibres of anterior roots of cauda equina; nervous system otherwise normal; cultures on agar from brain, medulla, and cord negative; no bacteria seen in tissues.

GROUP II (c).—*Cases in which Lesions were found in both Spinal Cord and Peripheral Nerves.*

Eisenlohr, *Deut. med. Woch.*, 1890, xxxviii, p. 841.	Female; 59 years; 17 days.	None.	Paralysis ascending from legs to medulla.	Pain in legs and back; diminished sensation.	Retention of urine.	Triceps reflex only, retained.	Reaction to faradaism in arms and intercostals; other muscles not tested.	Normal.	Pulmonary tuberculosis; spleen soft, not enlarged; anterior nerve roots degenerated; cord normal, except a focus of swollen axis cylinders and degenerative products in 11th and 12th dorsal segments; posterior roots normal. Bacteriological examination: 2 cultures, No. 1 occurring in all organs, *Staphylococcus pyogenes aureus*, No. 2 from spleen and sciatic nerve only, *Staphylococcus cereus* (Passet); sections contained a few bacilli and cocci, but no tubercle bacilli.
Centanni, *Ziegler's Beit.*, 1890, viii, p. 358.	Male; 7 days.	None.	First legs, later arms and diaphragm; dysphagia, paralysis of orbicularis palpebrarum; pupils normal.	Pain in legs and back.	Normal.	Absent.	Not tried.	T. normal.	Nerves: vessels prominent and surrounded by exudate; nerve fibres not much altered. Cord: leptomeningitis and inflammation around the central canal; atrophy of peripheral fibres; other parts of nervous system normal; in all peripheral nerves there is a bacillus in large numbers; cylindrical, with rounded ends, without spores or special grouping, usually found in endoneural lymph spaces, not found elsewhere; no cultures made.

References.	Sex, age, duration.	Ætiology.	Paralysis.	Sensation.	Sphincters.	Reflexes.	Electricity.	Pulse and temperature.	Autopsy.
Giuzetti, *Rif. Med.*, 1894, ii, pp. 5, 19, 31.	Male; 19 years; 9 days.	Exposure to wet.	Paresis; then paralysis of legs, then of arms; dysphagia, dysphonia, asphyxia.	Formication; pain in median and trigeminus trunks on pressure; some tenderness in muscles; pain over vertebral spines.	Retention.	Absent.	Not given.	P., 120. T., 99°.	Spleen large and soft; disintegration of fibres of cord most marked in lumbar region, absent in medulla; ganglion cells cloudy with indistinct nuclei; about vessels a ring of granular substance, not staining like fibrin; recent punctiform hæmorrhages in medulla and pons; degeneration of anterior and posterior spinal roots, most marked anteriorly. Peripheral nerves: multiplication of nuclei of neurilemma; some vessels of nerves surrounded by leucocytes. Bacteriological examination: Rabbit injected with emulsion of spinal cord and spleen pulp, result negative; cultures from brain, cord, sciatic nerve, heart's blood, spleen, mesenteric glands, urine, on various media, all negative except 2 tubes from sciatic and 1 tube from cord which grew a chromogenic bacillus; no bacteria seen in sections.

Ballet, *Méd. Week*, 1895, iii, 526.	Male; 33 years; 7 days.	Influenza.	Of legs, then of arms; dyspnœa, cyanosis.	Nearly normal.	Not mentioned.	Lost.	Not mentioned.	Fever.	Spinal cord: circumvascular changes, especially around the anterior horns; degeneration of nerve cells shown by Nissl's method; the anterior roots and peripheral nerves were "the seat of lesions without special features." Cultures from brain and medulla were negative; no bacteria seen in tissues.

GROUP III.—*Cases hitherto described as Acute Anterior Poliomyelitis, or Infantile Spinal Paralysis.*

Rissler, *Nord. Méd. Arkiv.*, 1888, xx, p. 22.	Child; 4½ years; 6 days.	Epidemic in Stockholm.	First of left leg, then of right; arms free; right pupil larger than left.	Pain in legs; sensibility normal.	Bladder normal; constipation.	Absent.	Not given.	Not given.	Spleen soft. Spinal cord: degeneration of ganglion cells; nerve fibres of gray matter and anterior columns of white matter are degenerated; proliferation of glia cells; occasional hæmorrhages; vessels increased in size and number; circumvascular infiltration; vascular changes almost entirely limited to gray matter; degeneration of anterior roots; degeneration of both hypoglossi and one vagus.

References.	Sex, age, duration.	Ætiology.	Paralysis.	Sensation.	Sphincters.	Reflexes.	Electricity.	Pulse and temperature.	Autopsy.
Rissler, *loc. cit.*	Female; 21 years; 8 days.	Epidemic (exposure to cold).	First of right, then of left leg; then of arms and trunk; dysphonia, asphyxia.	Normal; no pain.	Normal.	Absent.	Not mentioned.	T, 100·5°.	Spleen soft; changes in cord similar to previous case; medulla not examined.
Rissler, *loc. cit.*	Child; 5 months; 6 days.	Epidemic.	Paralysis of all four extremities; dysphagia, dysphonia, asphyxia.	Test impossible.	Normal.	Lost.	Not tried.	Not given.	Spleen soft; changes in cord same as in other cases; lesion in vagus.
Dauber, *Deutsch. Zeitsch. f. Nervenh.*, 1893, iv, p. 200.	Boy; 8½ months; 5 days.	None.	Both legs and left arm; paresis of right arm; drooping of left labio-nasal fold; pupils react slowly; eyes turned upward and outward; dyspnœa.	Not stated.	Not stated.	Absent.	Not stated.	P. rapid. T., 100° to 100·5°.	Medulla, intense hyperæmia around motor nuclei; circumvascular infiltration of round cells, which also form a free exudate in glia tissue; degeneration of nerve cells of nuclei, xii and x; central canal normal; changes chiefly in gray matter, though also in white; pia infiltrated. Spinal cord: similar changes in entire cord (cervical, lumbar); involvement of posterior horns as well as anterior; degeneration of columns of Clarke; lesion also seen occasionally in pyramidal tracts.

Goldscheider, *Zeit. für klin. Med.*, 1893, xxiii, p. 494.	Female; 2½ years; 12 days.	None.	Legs; dyspnœa, asphyxia.	Not mentioned.	Not mentioned.	Not mentioned.	Not mentioned.	Fever.	Degeneration of ganglion cells; vessels full; circumvascular infiltration; changes chiefly in anterior horns, though also in lateral columns; pia infiltrated, most markedly in lumbar region, also in cervical and dorsal; medulla, nerves, muscles not examined.
Redlich, *Wien. klin. Woch.*, 1894, No. 16, p. 287.	Child; 5 months; 10 days.	None.	Four extremities; dyspnœa, dysphagia, dysphonia, asphyxia.	Slightly diminished sensibility.	Not given.	Lost.	No reaction.	Slight fever.	Poliomyelitis of whole cord, extending into white matter; acute degeneration of ganglion cells; circumvascular infiltration of vessels; anterior horns most affected; areas of inflammation in medulla, especially around nuclei of 12th and 10th nerves. Nerves: some degeneration in brachial plexus.

SYMPTOMATOLOGY.

LESION.	None found.	In cord.	In nerves.	In cord and nerves.	Infantile spinal paralysis.	Total.
Group	I.	II (a).	II (b).	II (c).	III.	
Age:						
Adults	15	12	3	3	1	34
Children	1	0	1	1	5	8
Not given	0	2	0	0	0	2
Sex:						
Males	13	8	4	3	1	29
Females	3	4	0	1	2	10
Not given	0	2	0	0	3	5
Duration:						
Length in days	6–28	3–28	5–12	7–17	5–12	3–28
Ætiology:						
None	8	10	3	2	3	26
Phthisis	1	0	0	0	0	1
Variola	1	1	0	0	0	2
Indefinite infection	2	1	1	1	0	5
Exposure and exertion	2	0	0	1	0	3
Epidemic	0	0	0	0	3	3
Typhoid fever	1	1	0	0	0	2
Syphilis	0	1	0	0	0	1
Diphtheria	1	0	0	0	0	1
Sensation:						
Normal or nearly normal	8	12	2	1	3	26
Pointing to neuritis	7	1	2	3	0	13
Not given	1	1	0	0	3	5
Sphincters:						
Normal	7	8	1	1	3	20
Involved	4	4	2	2	0	12
Not given	5	2	1	1	3	12
Reflexes:						
Normal	2	0	0	0	0	2
Diminished	1	1	0	0	0	2
Lost	12	8	2	4	5	31
Not given	1	5	2	0	1	9
Electrical Reactions:						
Retained	10	5	3	1	0	19
Absent	0	0	0	0	1	1
Not mentioned	6	9	1	3	5	24
Temperature:						
Normal	8	6	3	3	0	20
Moderate fever	5	3	1	1	4	14
Complicated	1	2	0	0	0	3
Not given	2	3	0	0	2	7

Thus it appears from the forty-two cases cited that fatal acute ascending paralysis may be associated with any one of four conditions, which may be classed as follows:

1. Cases in which no histological changes were demonstrated in the nervous system.

2. Cases in which there was an acute exudative inflammation of the cord and medulla, and sometimes of the brain.

3. Cases in which there was an acute inflammation of the peripheral nerves.

4. Cases in which there was an acute inflammation both of the central and peripheral nervous systems.

1. *No Changes Found.*—After careful consideration of the cases in which no lesion was found in the nervous system, we consider it a matter of considerable doubt whether this same negative result would have been reached if the present accepted methods of examination had been applied. In six of the sixteen cases in Group I the peripheral nerves were not mentioned or not examined. In only two is it stated that the nerves were examined in osmic acid. Furthermore, in none of these cases were the most improved methods for the study of cytological detail employed. It is entirely possible that Nissl's stain might have revealed lesions in the ganglion cells which, examined by cruder methods, failed of demonstration. In spite of these objections to the methods used, the examinations were conducted by skilled investigators, and a long series of examinations, with uniformly positive results, will be necessary before it can be postulated that fatal acute ascending motor paralysis can not exist without demonstrable changes in the nervous system.

2. *Changes in the Cord only.*—Of the fourteen cases from Group II, (a), in which well-marked changes were found in the cord alone, in nine the peripheral nerves were not examined; in only one of these were the clinical symptoms of neuritis present. The remaining five cases conclusively demonstrate that a lesion limited to the cerebro-spinal axis may produce acute ascending paralysis. Moreover, here must be added four of the cases reported as anterior poliomyelitis, in which the pathological changes were the same as those found in the cases of Group II, (a), and in which the symptoms were typical of acute ascending paralysis. There is, there-

fore, a total of eighteen cases in which distinct lesions were found in the cerebro-spinal axis alone.

3. *Nerves only Affected.*—The changes of peripheral neuritis alone were satisfactorily demonstrated in one of these four cases in Group II, (b). In Hun's case degeneration of nerve fibres was found in the anterior roots of the cauda equina only. Such a lesion being entirely inadequate to cause death, this case might equally well have been placed with those in which no lesion was found. It is impossible to say from Putnam's report whether or not the cord was involved as well as the nerves, as it is not stated in his article that the cord was examined. Thus there is only one case from Group II, (b), to show that a lesion limited to the peripheral nerves may cause typical symptoms of fatal acute ascending paralysis.

Notwithstanding the contrary conclusions reached by some other observers, our plan of classification, necessitating a complete clinical adherence to acute ascending motor paralysis and a satisfactory pathological examination, has prevented us from including any further cases in this category.

If these cases of peripheral neuritis are to be associated with those of anterior poliomyelitis under the term of Landry's paralysis, such a classification can only be justifiable by the modern conception of the neuron.

With only a single element, the secondary motorial neuron between the cortex and the anterior horns of the spinal cord, and the primary motorial neuron between the anterior horns and the periphery, the distinction between peripheral and central changes of the nervous system has become less sharply definable.

So the cases of Landry's paralysis, in which pathological changes are found chiefly in the nerve trunks, may be explained by the assumption that the effects of the disease were chiefly in the primary motorial neuron; and, although the irritation acted originally on the protoplasmic portion of the neuron, those portions of the neuraxons farthest removed from the nutrient proto-

plasm showed the most evident results of the action of the toxic agent.

The same explanation may hold good for the primary motorial neuron, although no cases have as yet been examined with sufficiently delicate methods (e. g., Marchi's) to detect degenerations in the neuraxons of the cortical cells.

4. *Changes in Cord and Nerves.*—Only three of the cases from Group II, (c), can with complete accuracy be placed in this class; but, from the fact that in Centanni's case bacilli are reported in considerable numbers in the nerve sheaths (a most unusual occurrence), we have thought best to classify that case among those showing changes in both cord and nerves. To this class also rightly belong one case reported by Rissler and one by Redlich, detailed in Group III, and originally described as acute anterior poliomyelitis. In these the symptoms were typical of acute ascending paralysis, and pathological changes were found both in the cerebro-spinal axis and in the peripheral nerves.

It will be seen from the foregoing summary that there is a total of twenty-eight recorded cases of Landry's paralysis in which lesions were demonstrated either in the central nervous system alone, or in the central and peripheral nervous system.

But few bacteriological examinations in any of the preceding cases have been made. Eisenlohr (Group II, (c)) found the *Staphylococcus pyogenes aureus* in all organs, and the *Staphylococcus cereus* (Passet) in the spleen and sciatic nerve. Curschmann found bacilli in the white substance of the cord, which he professes to have demonstrated (by inoculations in rabbits) to be the typhoid bacillus. Giusetti cultivated a chromogenic bacillus from the cord and sciatic nerve. Centanni saw small bacilli in the sheaths of the peripheral nerves. Oettinger and Marinesco demonstrated cocci in the ganglion cells, in leucocytes, and in the tissues around the central canal. Remlinger isolated, with careful technics, a pure culture of *Streptococcus longus* from the spinal cord. Inoculation of rabbits from the tubes was without

effect. Partial bacteriological examinations in four other cases were negative.

Although conclusive demonstration is wanting, a consideration of the ætiology, symptoms, and pathology of this disease points toward the action in the body of some toxic agent, in many if not in all cases of bacterial origin, as the direct cause of the lesion. In support of this belief we may refer to the occurrence of lesions identical with some of those described above in epidemics of anterior poliomyelitis (Medin, *Verhand. d. X. inter. Cong.*, vol. ii, 6, 37; Cordier, *Lyon méd.*, 1888, lvii; Caverly, *Am. Med. Jour.*, 1896, xxvi, p. 1). Also Bristowe and Horsley (*Brit. Med. Jour.*, 1888, vol. ii, p. 1110) report a case of typical ascending paralysis, without detailed autopsy, due to undoubted rabies.

Finally, Thoinot and Masseline (*Rev. de méd.*, June, 1884) have succeeded in producing symptoms of spinal paralysis in rabbits by intravenous injections of *Bacillus coli communis* and of *Staphylococcus pyogenes aureus.*

The lesions, as detailed in the foregoing tables, are those of an intense acute exudative inflammation of a character such as is usually dependent on a bacterial cause. The condition of the abdominal viscera points distinctly to an infectious element in the local process. If we are to accept the negative pathological reports collected in Group I, the symptoms in such cases may be most reasonably explained by the presence in the circulation, and especially in the region of the central nervous system, of a toxic agent which destroys nervous function without causing demonstrable histological lesion.

From the limited data it has been impossible for us to distinguish any differential clinical signs from the various classes. While most cases associated with extensive pathological changes bore the clinical stamp of infectious disease, in some with negative pathological report this feature seemed equally pronounced. Again, sensory changes indicative of neuritis were present in cases in which the nerves were found to be normal, as well as in those in which neuritis was demonstrated.

Marked febrile disturbance was not prominent in any of the cases, but occurred in isolated instances in all classes.

The application of electrical tests seemed of no assistance in determining the pathological condition.

While it is not to be expected that the bladder and rectum should be paralyzed in lesions limited to the peripheral nerves, these functions may equally well remain unaffected if the lesions of anterior poliomyelitis are but slightly marked in the sacral region. Other clinical data were equally lacking in differential value. In the table on symptomatology may be found a review of the symptoms as they occurred in the various groups.

The conclusions from the present study may be summarized as follows:

Acute ascending paralysis is an acute toxæmia in which the poisonous agent affects chiefly the nervous system.

The commonest seat of the lesion is in the spinal cord and in the medulla, and it may be present in the cortex and in the nerve roots. When in the spinal cord the lesion is that of acute anterior poliomyelitis—namely, an acute exudative inflammation, following the distribution of the central branch of the anterior spinal artery, with cellular infiltration of the circumvascular sheaths, degeneration of ganglion cells, loss of structural elements, and with or without degeneration of the anterior roots. The lesions in other parts of the cerebro-spinal axis are of a similar nature.

The evidence that the lesion of typical Landry's paralysis may exist in the peripheral nerves alone is based upon a single case reported twenty years ago by Dejerine. When the lesion affects the peripheral nerves there are increase of neuroglia cells and degeneration of nerve fibres.

It is at present impossible to deny that acute ascending paralysis may run a fatal course without leaving demonstrable histological changes in the nervous system. It is certain that the cases with negative pathological report did not present the marked vascular lesions of acute

anterior poliomyelitis, but it seems probable that in these cases there were changes in the ganglion cells demonstrable by delicate methods. From the present data it seems to us impossible to distinguish by the clinical symptoms the different types of lesions.

The investigation of the present case was conducted in the pathological laboratory of the College of Physicians and Surgeons. For kind revision of the paper we are greatly indebted to Professor T. Mitchell Prudden and to Professor M. Allen Starr. Our thanks are due to Dr. Hodenpyl, pathologist to Roosevelt Hospital, for the opportunity of reporting the case.